Longevity technology:

Longevity gum, “More than 100,000 tons of chewing gum being consumed every year.”, of cavity preventing xylitol gum, “dentists from all around the world recommend daily ingestion of up to 5g of xylitol (around 9 mints or 3-5 pieces of gum per day” So if people chew five pieces of gum per day, 1825 pieces a year; 730 million or 1 billion gum chewers is plausible at a little over 10% of 2019 global population, and at 1825 pieces a year that is a little less than 2 trillion sticks of gum a year, at 20 pieces of gum each 24 hours per chewer that is 8 trillion pieces of gum. At a one cent premium per piece of longevity wellness gum that represents 80 billion us$ annual revnue from longevity and wellness increasing chewing gum.

AEDG chewing gum, also if a levo and dextro amino acid version of AEDG is found to be longevizing and wellness causing then AEDG could reach the GI tract for absorption.

Lithium at gum: Lithium in the water supply is correlated with people living longer, and makes laboratory nonvertabrates live 9-20% longer. If a flavor neutral or yummy lithium gum additive can be found, which could possibly arise from linking a 10,000 times sweeter than sugar sweetness peptide to lithium at a stomach dissolvable lithium chelator molecule, then at 5 sticks a day, and 5 mg of lithium per day, then 1.001 to 70 mg of delicious combined lithium/sweetness peptide/chelator molecule would be at each stick of gum.

Also, they can put anything, at perahps 200 mg at liquid center gums like chewels.

Some chewing gums may already have a peptide/peptone/protein component. calcium casein peptone is a texturizer at chewing gum, “at a use level up to 5% wt/wt”

Production of beneficial drug peptides from milk (casein) and grain (gluten): Modified proteases like trypsin could make different digestion products, some of which are drugs. Genetically engineered organisms would make the new proteolytic enzymes with customized products, which would then turn other things into beneficial medical peptides affordably. This brings Genetically engineered production’s product affordability to natural products.

Even more affordable than engineered enzymes: plants that make modified gluten with trypsin or pesin dividing regions that repeat, with a peptide of value between them: Also, as plants produce glutens, it is possible that producing Genetically engineered organisms with the sequence (trypsin or pepsin cleavable location) -**peptide of value**-(trypsin or pepsin cleavable location): as the peptide-of-value-polyrepeat at genetically modified gluten could produce a highly affordable source of beneficial peptides.

Modifications of gluten to produce valued peptides released from digestion with ultraffordable already available bulk trypsin or pepsin:As a system the technologist would just swap in the amino acid sequence of interest at the **-peptide of value-**

location at the genome. During about 2005 AD the barley lab I worked at would get about 3 successfully genetically engineered plants for every 100 prepared (embryo sliced, soaked in transfection liquid, agar placed) barley embryos suggesting immediate rapid production of over 200 different versions of gluten with different -peptide-of-value- per researcher per year using very simple technology I was able to use with 1 hour of training; the technology I used likely has transfection efficiency and hourly production with new variant protocols that are 10 or 100 or even 1000 times more productive from automated slicing and multiwell plates.

It could be nifty if taking a protein with amino acid (peptide-like) sequences that already have bridges (like S

bridges or different bridges) and then putting something like a trypsin or pepsin-division sequence at the four corners of the bridge :-:, would then produce a variety of different customizable bridged peptides easily and reliably; they could even make an enzymatically attachable spacer amino acid string of genetically variable length to place between the two bridge sides like n or n that would predictably provide the right distancing of briding amino acids to favor amino-acid bridging. This likely already exists.

Can a trypsin or pepsin (notably a chemical variant that does not interact with the protein source, like milk, until the hydrogen ions in the stomach modify the trypsin molecule) be swallowed with a food, like a trypsin or pepsin milkshake, to

produce a biologically active peptide in the stomach or even other parts of the GI tract?

Casein, the 80% of milk's protein protein is processed like, "manufacturers combine casein with calcium hydroxide at high alkaline levels and dry the protein" **So could a non pH/pOH molecule like a carbonium ion, a methyl ion, or an ammonium ion make a novel protein chemical, that possibly with enzymatic digestion becomes a beneficial drug**; sort of like casein with ammonium makes a bunch of c=c-c=c peptides that have ammoniums on them, thus looking sort of like metformin, a biguanide with numerous c=c, That goes with preconcentration, predigestion protein sources with lots of gaunidine could produce metformin function-similars

with ammonium ion (pNH3/pNH2) treatment. This could also be used on digests of the protein gluten.

At casein as well as gluten they could screen every n sized group of peptides available from a library of possible published custom digestions of the protein (producing like all 7 mers, all 40 mers etc) against activity databases to see if any of them are drugs, they could also massively parallel make molecular receptor attachment models of some amount of casein's N possible truncated peptide constituents to find new drugs that could be made from casein or gluten.

Opiod peptide from digestion of milk: a 3 (H-Tyr-Pro-Phe-OH) ,4,5,6, or seven-amino peptide (H-Tyr-Pro-Phe-Pro-Gly-Pro-Ile-OH) like beta-casomorphin-7; perhaps some opiod

peptides are actually enjoyable, which are also minimally harmful, perhaps from localizing at only particular brain regions like the nucleus accumbens; **It is possible that nonCNS opiod peptides relieve discomfort without having cognitive or behavioral effects.** I perceive just putting a hydrophilic lipophobic length of, or external hydrophilic or lipophobic tertiary structure outer layer of amino acids or a polyglycine length on a peptide will keep it on the body side of the blood brain barrier, so that could be a thing that relieves discomfort or could aslo provide anesthesia.

A map, possibly constructed with positron emission tomography, or other approaches, of a screened library of which peptides concentrate at what brain regions, as well as what

body regions, and an immunocolorization map (or niftier technique) of peptide localization at each cytotype and tissue type would be beneficial to the creation of beneficial drugs, notably those producible from gluten digests and probiotics and gene therapy as well as possible germline modification. Nootropic: numeous nootropic peptides are described online and at the scientific literature, https://ultranootropic.com/ ,would vasopressin, thymosin beta 4, semax, noopept, as well as many other peptide variants that only localizes at the frontal lobes improve cognition and memory and other cognitive things without effecting emotional capability (limbic areas) or bodyside functions (cerebellum, brainstem)?

Noting CNS effects on longevity,

screening all nootropic peptides to see if any of them are also longevity producing peptides could find new longevity drugs, as well as amino acid sequences that van be function mimiced with peptide mimetic drugs to produce completely new lifespan lengthening drugs.

also, “Casein peptides are used for high blood pressure, high cholesterol, anxiety, fatigue, epilepsy, intestinal disorders, cancer prevention, and stress reduction”

Could casein be used as an ion transporter to different cytotypes?

polyprotic acid, or a polyhydroxyl base

s from changing something like “sodium (salt of) Also, they can put anything, at perhaps 200 mg at liquid center gums like chewels.

Do any oligosaccharides (like sugar mini-polymers) have drug effects? Are any of them longevity or wellness effects like polyribose might have, polyribose molecularly sapced NR or NMN that turns to NMN at cytoplasm, as a possible enzymatic or some benefit to the brain as a food, or fostering beneficial probiotic growth,

Previously described are possible artificial colors that heighten wellness or longevity to be used as food additives. c=c-c=c-c=c structures are frequent at some colorizing chemicals. Also, a longevity version of bright yellow B vitamins could be

possible.

Microsugar lancets like at applique needlesless drug delivery could have some activity at gum. That suggests a pack of gum could immunize against atherosclerosis, perhaps a dose per decade, or even a dose per century.

A month of gum chewing with highly localized, less than than mentally perceptible effects on feeling normal, senolytic could be a one month longevity treatment.

Although candy with immunoactive material at microlancets could also do immunizations, it is possible swallowing immunobeneficial or other longevity technology gum could be beneficial.

AEDG linked to carbohydrates; lithium linked to carbohydrates, ribose linked

to AEDG could concentrate AEDG at the heart, providing benefit.

AEDG linked to lactate or lactic acid could concentrate at the brain, causing benefit; as a minute amount at food, gum, or candy, concentration of AEDG at the brain could provide benefit, notably though AEDG has something to do with pineal gland chemicals, so brain concentration could permit lower doses, be more likely to provide projected benefits, possible enhance or otherwise effect fertility (50% greater conception rate from melatonin at IVF)

NGF and BDNF heightening 2 amino acid peptide, noopept, “In animal studies, Noopept has been shown to stimulate the expression of two important cognition-related chemicals, Nerve Growth Factor (NGF) and Brain-Derived Neurotrophic Factor (BDNF).”

Could those two amino acids at noopept, proline glycine, or any other **nootropic peptide, be used when attached to other drugs** cause **high utility brain localization**, “Brain-Derived Neurotrophic Factor (BDNF) has a similar role to NGF but is primarily active
in the hippocampus, cortex, and basal forebrain, areas of the brain that are vital to learning, memory, and higher thinking”, also, “readily crosses the blood-brain barrier”, so attaching 2 mer (prolyl glycine) noopept to 3 mer opiate peptide (Tyr-Pro-Phe) making 5 mer pro-gly-tyr-pro-phe as a brain concentrating opiate peptide?

Proline-glycine could be an effective way to get numerous other drugs to pass the blood brain barrier, perhaps phenibut-Pro-Gly could work at orders

of magnitude less milligrams per dose, and it is also possibly Pro-Gly causes drugs to pass the ovary-blood barrier as well, improving fertility drugs and possibly making contraceptives even more affordable on a $/Kg manufacturing basis.

Protein Kinase RNA-Activated inhibitors, or PKR inhibitors like C16 are peptides described online as causing new path learning after one session as compared with several sessions for unmedicated mice; could PKRI be used to localize other drugs to the brain areas that cause the “one training session works as well as several” effect? CART peptide linked to PKRI is one possibility for a new nootropic that also makes learning with many fewer examples or less practice duration possible. Even

linking Pro-Gly (noopept) which increases BDNF and NGF to PKRI like C16 could cause those rapid-learning neural areas to grow causing lasting increases in intelligence. PRKIProGly can be constructed and ordered online.

Are there any peptides that pass through cartilige and joint tissues rapidly, these are different than "blood brain barrier", but if there is any preferential transport there might be peptide that does that at joints; chondrotoin, MSM, hyalonuric acid and others could all be linked to such a joint transport protein. This could also heighten migration of other drugs. This would treat or prevent some joint decay, causing more youthful joint form and usefulness.

Genetically engineering a plant

that treats schizophrenia and psychosis: There could be a peptide, findable at a library of less than 576 two mer peptides that specifically effecs 5HT receptors, Latuda which functions only at HT2 receptors and is absent effect on D1,D2, orther dopamine receptors, causing fewer side effects, could perhaps have a functionalike peptide, and then the peptide engineered into plants, brewing yeast, yogurt, vaginal probiotics, and oral probiotics, making antipsychotic medication grwoable and able to reach more than 70 million people globally. Of the Pro-Gly two amino acid noopept, online it says, “Noopept modulates the activity of both AMPA and NDMA receptors”, so they could **screen a combinatorial library of all the 2 unit peptide combinations of 24 different peptides, which is near**

576 different peptides on 8 mice each, to get p<.05 at behavior change, like nonpsychotic begavior, and immunocolorization mapping of the brains and bodyside nerves to see which peptides caused localization at dopamine neurons, much the way Pro-Gly concentrates at AMPA and NMDA neurons. Also, as serotonin neurons have many published activities these serotonin active or also serotonin neuron localizing neurons could have numerous beneficial drug effects.

Although noopept Pro-Gly is functional at 10mg oral dose, could a leve-dextro version of the amino acids make it omit being digested, causing a nootropic doasge in the micrograms?

Similarly, could a levo-dextro version of opiate peptides omit being digested thus causing microgram functional dosages? Also, do opiate peptides

work more enjoyably if snorted or vaginally applied, or made to be a buccal absorption alginate gel mouth coating?

A variety of ribosomal activity nootropics that might, or might not function like PKRI C16, Protein Kinase *RNA-Activated Inhibitors,*“What makes PKR inhibitors an EXTREME example is how it works. It essentially disables a security feature of the brain that helps to prevent viral infections” suggests the possibility that either genetic material reaching ribosomes omits a prescreening of some kind, or that the ribosomes work more rapidly, or that tRNA availability goes up,

Longevity technology: Dastinib with querectin are Senolytics that benefit the brain:

The senolytic: is described as, “Senolytic treatment of AD mice selectively removed senescent cells from the plaque environment, reduced neuroinflammation, lessened Aβ load, and ameliorated cognitive deficits. Our findings suggest a role for Aβ-induced OPC cell senescence in neuroinflammation and cognitive deficits in AD, and a potential therapeutic benefit of senolytic treatments. “dasatinib and quercetin (D + Q), can selectively eliminate senescent cells from pathological tissues”

At mice, the senolytic dose was 12 mg/Kg of dasatinib and 50 mg/Kg querecetin, oral gavage with PEG/saline vehicle; so perhaps 70 mg per day of D for a human, utilizing the 1/12 mouse dose thing, and 350 mg of querecetin per day. Dasatinib is

prescribed for 12 months or possibly longer as an anti-cancer drug, but the senolytic dosage duration at the mouse experiment is 9-10 days. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6605052/

Dasatinib causes mice on an 11 week long dose to be better at learning motion pathways, at (youngish) 3.5 month old mice, rigged to be messed up, “In peripheral organs, partial elimination of senescent cells (~30%) is sufficient to restore tissue homeostasis and function in disease models and during aging[7,29,30]. We next determined whether longterm intermittent senolytic treatment could ameliorate Aβ plaque pathology and/or improve cognition in APP/PS1 mice. Beginning at 3.5 months of age, female APP/PS1 AD mice were treated with either D +

Q or vehicle once weekly for 11 weeks (Fig. 5a). Hippocampus-dependent spatial learning and memory were evaluated by testing the mice in the Y maze immediately before, and at the 6- and 11-week treatment time points, and mice were also tested in the water maze during treatment week 10. Mice were euthanized at 11 weeks and their brains processed for biochemical (one hemisphere) and histological (the other hemisphere) analyses. [and then it says] Compared with vehicle-treated APP/PS1 AD mice, APP/ PS1 AD mice treated with D + Q performed significantly better in the Y maze at both the 6- and the 11-week time points (Fig. 5f). In the water maze tests, D + Q treatment enhanced memory acquisition (more rapid learning of the location of the hidden platform) and memory retention in the probe trial” Notably

at a graphic at the paper, dasatinib with querecetin caused large but then identical learning effects; at day 4 of cumulatively learning a pathway finding task the drugged mice were approximately 62% better learners, but at day 5 they were identical.

At a different paper, “senolytic therapies could be administered intermittently, serving to reduce the senescent cell burden by treating quarterly or even annually, which minimizes the risk of side effects”, “Treatment of mice with dasatinib plus quercetin (D + Q) improves cardiac ejection fraction and increases vascular reactivity in old mice after a single, 3 day treatment course [30,34]. In addition, D + Q treatment decreases vascular calcification and increases vascular reactivity in hypercholesterolemic, high fat diet fed

ApoE$^{-/-}$ mice after three monthly 3 day treatment courses”

They could see if a different chemotherapeutic drug, nilotinib, that works on the same kind of cancer as dastinib is a beneficial senolytic, possibly with nonoverlapping activity at different body cytes or body tissues.

Localization peptides or proteins attached to senolytics like dasatinib could cause even greater benefit. Pro-Gly could cause brain concentration, noting that dasatinib is published as heightening learning ability it is perhaps beneficial to have a senolytic reach the brain. Chondrotoin or MSM molecular moeity on senolytics could cause greater joint youthfulness function, reduce immunoreactivity and sequelae; the cytotypes senolytics remove are

published as linked to bone-joint illness, suggesting senolytics could produce younger phenotype function at bones and joints.

Fisetin is a senolytic, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6197652/ “The natural product fisetin has senotherapeutic activity in mice and in human tissues. Late life intervention was sufficient to yield a potent health benefit.” as well as, “chronic administration of fisetin to wild-type mice late in life improved tissue homeostasis, suppressed age-related pathology, and **extended median and maximum lifespan”, “**This result, similar to a recent report on the combination of D ± Q, is the first to document extension of both health span and lifespan by a senolytic with few side effects, even though administration was started

late in life.”,

Dose: “mice were fed Teklad 2020 chow (Envigo, Madison, WI) prepared with or without 500 ppm (500 mg/kg) of fisetin (Indofine Chemical Co., Hillsborough, NJ) by Envigo. Co. (Tampa, FL). For oral administration of fisetin, mice were dosed with 100 mg/kg of fisetin in 60% Phosal 50 PG:30% PEG400:10% ethanol or vehicle only by gavage.” That represents 7 grams of fisetin every 24 hours at a direct, non compensated for mouse size dose, or **583.3 mg every 24 hours at a compensated mouse dose**. At the fisetin placed at food, “diet with or without supplementation with 500 ppm (500 mg/kg) of fisetin, *ad libitum* (approximately 60 mg/kg fisetin per day). The mice were exposed to a fisetin diet intermittently from 6 to 8

then 12–14 wks of age” which could be communicating that each Kg of mouse (that is a lot of mice) got 60 mg of fisetin, so 4.2 grams per day, without mouse division number, or **350 mg per day** at the mouse dose equivalent convention number. On ebay, fisetin is $22 for 10g.

At the paper, 20 micromolar fisetin has twice the cytonumber reduction as 5 micromolar fisetin at cultured cytes treated for 48 hours, and the difference in messed up cytes goes from no effect at absence of drug to 1.1/.5, or about 55% reduction of messed up cytes at 20 micromolar.

A different graph displays fisetin at cultured cytes’ senolytic activity as 1.3/.4 or 69% reduction in senolytic cytes.

They measured the senolytic activity

of 11 different chemicals, fisetin at 69% was more effective than curcurmin as a senolytic at about 50% https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6197652/

It is possible that sequential or simulataneous senolytics could benefit survival and promotion of youthful cytotypes, so curcurmin with fisetin, or at a sequence with each other could be beneficial. **Taking them simultaneously could possibly work better** as it is possible the nonoptimal cytes express immunoattractants while processing senolytics, and doubling the amount of immunoattractants or simultaneously producing different kinds of immunoattractants could cause greater immune response that removes nonoptimal cytes.

Notable for fisetin in food compared

with once a day oral dosing, fisetin has a plasma half life, so keeping plasma levels high and steady could be beneficial as the food only had 350 mg in it, compared with the 583 mg in the once daily dose, I do not know if they had identical senolytic activity yet though.

If a human eats .5Kg of carbohydrates that is 2000 calories, and at mouse dose would have 250 mg of senolytic fisetin, suggesting the 583.3 mg a day dose is better, noting at cultured cytes doubling fisetin concentration doubled senolytic acivity. also, at a different paper dasatinib was more effective at higher doses.

An immunization could have one of the effects of senolytics, “Senescent cells can develop a

senescence-associated secretory phenotype (SASP), consisting of pro-inflammatory cytokines, chemokines, and extracellular matrix-degrading proteins", **immunizing against extracellular matrix-degrading proteins could cause greater tissue functionality longer, and senolytics are researched as causing greater wellness and younger phenotype**. extracellular material might be particularly reachable with antibodies. It is possible that as some immunizations last as long as a person, immunizing against extracellular matrix degrading proteins could be a one dose lifespan increasing and wellness increasing immunization, possibly functional at farther than postpubertal ages even when given to a child, during the period when people routinely get immunizations and parents highly

prioritize immunization activities and coverage. It is also possible that “matrix degrading proteins” have an effect on dermal cytes and structure, which I perceive I read have something to do with the word “matrix proteins”, that causes this possible longevity wellness immunization to also be a beautification and youthful appearance sustaining possibly one dose treatment, heightening popularity and voluntary use. The other chemicals mentioned, “pro-inflammatory cytokines, chemokines” could also be immunized against to benefit longevity, wellness, and phenotypic youthfulness; I pereive there are a number of these cytokines and chemokines, so a list of which have the highest mass/volume concentration both at the circulatory system and outside, right next to the cytomembrane at the intracyte space,

and then immunizing against those, could concentrate effectiveness although cytotype and tissue specificity could make immunizing against all of them notably more effective; One benefit of immunizing against a multi digit (2 digit? three digit?) number of chemokines and cytokines is that some of them may have multidecade effective immunization coverage while others might have different immunization coverage, This partial yet long duration removal of deleterious cytokines and chemokines and matrix protein harming chemicals could actually interrupt and derail any harmful processes where the deletarious cytokines, chemokines and matrix-messing up chemicals effect each other, build on each other, or saturate body repairs, from combining or cascading each other’s

effects. A person that is good at math and algorithms could look at a biochemical network, then come up with a minimum number of items to remove, at certain chemical-link distance from each other, to break a network effect, cause a different group average, or modify the persistence of an emergent effect, sort of like whats the fewest steps to wipe out a deleterious eignevalue? Immunizing against that group of chemokines, cytokines or matrix-protein messer-uppers could do that math, causing greater longevity, wellness, healthspan, and youthfulness of phenotype.

Protein or peptide linked drugs could traverse vascular plaque cytopiles to deliver beneficial drugs that shrink of remove vascular plaque blobs and

cytopiles and be a cardiovascular wellness drug, “[senolytic]reduces senescent cell-like, intimal foam cell/macrophages in vascular plaques”; they could screen the 526 peptide library of 2 unit (mer) peptides formed from 24 amino acids to find out if any cause preferential traversal of atherosclerotic plaque cytopiles and possibly cholesterol coatings. lipophilic peptides could be better at traversing cholesterol coated or layered cytopiles. lipophilic peptides linked to physiologically harmless immunocyte attractants could cause macrophages and WBCs to find atherosclerotic plaque particularly attractive to glom and remove. Making a protein digest of bacterial cytowalls, then linking them to lipophilic peptides could cause the immune system’s rapid and effective response to bacteria to be directed at

atherosclerotic plaque, causing greater wellness and possibly improving cognition with improved CNS circulation, as well as reducing risk of cardiovascular disease. Immunizing against cholesterol works on rabbits to reduce cardiovascular nonoptimality, immunizing against plaque blobs could remove them: also just as there are many different velocities and “brush strokes” to cleaning off a nonbiological item, different velocities of immunoresponse as well as different biochemicals to remove (immunoglom) first could be tested to find the immunocleanup of vascular plaque blobs that was most beneficial and with the least risk.

a novel senolytic mechanism with a “pick preferred candy out of a pile before getting on a school bus”

metaphor: Some chemical transport channels of the exteriors of cytomembranes are possibly durably pluggable with molecules, these molecules are also consructed to have a lengthy molecular tail that the immune system recognizes and is reactive to. It is possible that deleterious cytes like the kind senolytics remove, as well as scar or encapsulation tissue cytes, as well as piled up cytoblobs at vascular arterial plaques, have greater molecular transport of some specific chemical. Then, I perceive that when a person gets a harmless viral cold, that they get symptoms for 3 days before the symptoms get better, that suggests that a 2019 AD human’s immunse system takes 72 hours to produce the antibodies to remove an infection.

Labelling many things, then using a

gentle wash to provide unwell/well cytocontrast: Decorative plugs that occupy a transport channel at the cytomembrane diffuse away, with a half-diffused amount at 24 hours, so 96 hours after the labelling dose, only 1/8 as many decorated plugs are on cytes. If the deleterious cytes stared with 80 plugs per cyte they will still have 10, but the cytes that only had 6-8 decorater plugs will have about 1 or zero.

Noting that senolytics increase longevity, wellness, healthspan, and cause younger cytophenotype and tissue phenotype: **a new kind of senolytic**: Have the person eat or be dosed with an immunoreactive chemical that has a cytomembrane molecular transport channel plug that is like 3 to 40 times more likely to plug up a molecular transport channel at

the external cytomembrane and accumulate at a deleterious cyte like the kind of cytes senolytics terminate. Noting well cytes and deleterious cytes differ as to their transport channels (senolytics are previously published as reaching their goals preferentially) Give them a big enough dose so that each deleterious cyte gets say 80 antibody-reactive plugs for every 2 at a well cyte, or even 90 antibody reactive plugs at a deleterious cyte for every 30 at a well cyte. Then give the human a big dose of the decoration (the antigen without the plug part) to activate the immune response, doing this after 96 hours of gentle diffusion washing away some of the decorator plugs, the big dose of plugless decoration causes a large immunogen response, after most of the well cytes are absent decoration while the deleterious cytes

are still immunoreactive.

Also, along with the activation dose of decorator (absent plug) antigen to produce an immune response after **well cytes** immunoreactivity is decreased, It is possible to use the body’s apparently ordinary 2019 AD 72 hours to make an immune response to a viral cold effect, then have that 72 hour spontaneous immune response glom to and remove the decorative plug labelled cytes once 96 hours have progressed, the body has produced antibodies after the 72 hour viral cold-like process, and well cytes have only zero to perhaps 3 decorated plugs compared with 8 at a deleterious cyte. The deleterious cytes have been senolytically removed, and could even possibly be removed on a “senescent cytes have different transport channels” basis,

the thing that makes it a new senolytic is that it terminates cytes like a senolytic without an external drug toxin; similarly this could be a way to treat cancer, removing some of the oncocytes. Noting that some mechanism at senolytic chemotherapeutic drugs like dasatinib get preferential effect (thus likely membrane transport) at senescent cytes, and then the senolytic drugs terminate the senescent cytes, it is scientifically sensible to think senescent cytes have different molecular transport channels, or quantities of ordinary channels.

Plugging the transport channel while attracting immunoreaction, when the immunoreaction can be times or sequenced creates kind of algorithmic numerical

advantage in removing deleterious cytes. Possibly a plug that resides for just hours or 96 hours would preferentially remove deleterious cytes while using up all the circulating beginning antibodies to the decorated plugs; The decorator-plug immunoreactive cytes can use up all the circulating antibodies, that lack of antibodies causes the immunoreactions effect on well tissue to be minimized, which maintains organism well being. At some numerical versions, noting each deleterious cyte has more than 8 times as many decorative plugs on it while the well tissue only has between zero and 1/8th the sites and immunoreaction, After 8-24 hours after the 72 hours before the body makes its own antibodies to the plugs, at that 96 hour chronoregion, all the existing amounts of anti-plug

antibodies are utilized and there are near zero circulating antibodies, so there is an absence of further antibody glomming action on well cytes and tissues even though the immune system has been activated, activated with the same effective intensity as a response to a viral cold.

So like 100 milligram of antibodies gets used up on the highly 8:1 decorated transport channel-blocking molecules, while well cytes have a graphical distribution displaying the number of glommable plugs as centering on just one or even zero; It would take 24 hours to make another entire 100 milligrams of antibodies, so noting the decorated plug molecules wear off before that 24 hours while the body is producing the next 100 mg, the 24 hours that pass make cause just 1/4 of 1/8th the decorative

plugs to be at well cytes. The well cytes only get 1/32nd the immunoreaction cleaning dose of immune system response. Meanwhile the deleterious cytes still have a plurality of decorative plugs at each cyte, labelling them for glomming and removal.

Longevity technology:

So is there a sequencable series of natural allergens, or even easy to find and get harmless colds that can be screened, like a screenable library, to do this glom at the places beneficial senolytics are active, gently wash well cytes to experience immunoharmlessness, and also up immunocytes purposefully to terminate the labelled deleterious cytes? That would be a sequence made from preexisting viruses and

allergens at the 2019 AD typical population, where a human body proceeding through that sequence actually gets greater longevity, wellness, healthspan, and youthful phenotype.

So, uh, BCG, and possibly, MAO-B receptors on blood cyte surfaces, and perhaps there is something natural that occurs at capillary epithelial cytes; could blenderized pollen, perhaps with oil to make GI tract passaging liposomes, cause immunoreaction at the circulatory system? Also what about things like mushrooms, fugu, and other species immunoreaction physiological products? **Are there things like blenderized e. coli variations of particular kinds that cause varied immune response, which can be bred or engineered to be**

longevity, healthspan, wellnes,, and youthfulness of phenotype beneficial senolytics, because they have different lipopolysaccharides on their membranes?

Find the receptors or molecule transport channels that are at the exterior cytomembrane of senescent cytes, the kind longevity, wellness, healthspan and youthful phenotype producing senolytics effect.
Optimally find external cytomembrane molecular biology characteristic structures, senescent cyte-only receptors and molecule transport channels unique to senescent cytes.
Then gather a bunch of those unique molecular biology object-features, like proteins, molecule transport channel membrane protein structures, or

possibly lipid raft like cytomembrane fragments with a stable, typical to living structures of a molecular biological form of the kind of deleterious cyte terminating senolytics terminate, Then screen libraries of wild-type bacteria, fungi, plants, and even virus products to find out if they effect, modify, block, cause hypertransport at, or disintegrate these molecular biology structures.

Also, a novel thing, place a bunch of the actual molecules and proteins that are the unique biomolecular structures of the kinds of cytes senolytics terminate, at a culture medium, perhaps a (1000 times 1000) million multiwell plate, that contains a wide survey of bacteria, wild yeast, and variations on what humans think of as beneficial bacteria (probiotics); possibly use micropositioning to place

a zone of molecular biology unique structures, a diffusion gap. and then a bacterial growth area: this causes material that diffuses across the zone to be measurable as to its quantitative effect on the unique molecular biological structures of the kind of cytes senolytics direct their effects at; When some of the million sample multiwell plates find bacteria, fungi, even viruses hosted by human tissue culture cytes products’ or other materials that cause plugging, blocking, hypertransport amplification, or disintegration of the unique molecular biological structures of the kind of cytes senolytics terminate, then organisms that produce new senolytics have been identified and can be quantified as longevity, wellness, healthspan, and youthful phenotype producing drugs. These wild type organisms can also be bred

to have much higher amounts of senolytic chemicals. That is a new source of senolytic drugs.

As a technology, an immune response generating functions-like-a-senolytic yeast beverage or yogurt, plant, or plant pollen, bscteria, or even physiologically and attentionally nonperceptible dermal bacteria, similar to a probiotic, that is a senolytic from immunofunctioning, that is the immunosensitizing bacteria, plant, fungi or even virus causes greater longevity, wellness, healthspan, and youthfulness of phenotype like senolytics do. The humans, that is person's or people's immune response to optimally, the organisms or ok, but less nifty as it goes with actual human sustained production of concentrates, organism material concentrates, where both

and either of these causes the immune system to be sensitized to glom and terminate the kind of cytes senolytics terminate.

So, perhaps: blenderized e coli membranes, combined with cytomembrane molecule transport channel plugs that preferentially populate deleterious chemokine and deletarious cytokine **export channel (efflux) transport channels**, also combined with some oil to make liposomes that make it through the GI tract;

So where do the at-wild, at 2019AD society, efflux blocker molecules come from? Organisms at a culture medium with chemical travel zone and an area of chemically changeable molecular biology structures to measure the modification efficacy of each of the organisms forms is

described. As another source of efflux channel blocking molecules that are combinable with immunosensitizing decorations on the efflux blocking molecule, for immunoglomming and removal, which has a senolytic function effect.

It seems like it would be possible to screen a library of naturally occuring materials and molecules,proteins, peptides,andother things, as well as genetically engineered materials like new or enhanced proteins and peptides, to find out if there are any that plug the efflux channels that are effluxing deleterious biochemicals.

It might be that to a molecular biologist this is sort of easy, like “the transport channel is n angstroms wide and w angstroms tall. Anything hydrophobic/hydrophilic you make with an angle bend, a beta sheet or an

alpha helix on it will block the efflux channel of that size and form. If you make a 20 amino acid peptide tail and put iton the plugging protein, you can use an amino acid that is absent any environemental or body-wide previous immune sytem antigen stimulation, or immune system activity.
Immunocytes that react to the unique 20 mer peptide, after the “immune system alerting dose” are unlikely to react to any other physiological biochemical, minimizing immunological reactions drift, thus keepin nearer optimal human, immunofunction, human longevity, human wellness, and youthful phenotype form at the human.

The technology is that if just those cytes are immunoterminated which have a bunch of efflux channels where deleterious cytokines, chemokines,

and matrix protein messer-uppers are effluxed, then that immunotermination functions like a senolytic:

Gene therapy, that transfects and terminates deleterious cytes with something like apoptosis, gene therapy that produces immunoequivalents to senolytic drugs, or gene therapy that actually produces proteins or peptides that are senolytic in their own right. Technology of senolytic longevity, wellness, healthspan, and phenotypic youthfulness of form are developing. At 2019 AD, at any deleterious cyte where a senolytic accumulates at but previously did not terminate as it would more optimally do, gene therapy could be a way to get the 30% that fisetin does not reach, the 50% that curcurmin does not reach, or

the numerous kinds of cytotypes and tissues that any known senolytic might not reach the cytoplasm of (cartilidge, eye lenses, osteocytes, other nonvascularized tissues).

Curcurmin and fisetin are published senolytics; could curcurmin as well as fisetin molecules with an antibody alerting moeity or tail, attached with: an enzyme endogenous to the cytoplasm that divides the curcurmin or fisetin from the immune system alerting moeity, which then travels to the outer surface of the exterior cytomembrane, which alerts the immune system to terminate the cyte that the senolytic has already localized and concentrated at. That only works if fisetin, curcurmin, andother senolytics actually localize to deleterious cytes. If they just go to allcytes, but only terminate

deleterious ones, then a different technology would be the thing to make. If senolytics do localize and concentrate at deleterious cytes then putting immunoactivators on them would cause them to be even more senolytic as the immunocytes seek them out.

wellness technology: somehow leukocytes, macrophages and WBCs notice they are more effective at being an active and beneficial immune system when they travel past the capillary epithelia to actual cytotypes that provide a utility definition to a tissue (chondrocytes, pneumocytes, cardiocytes, dermatocytes, beneficial only immune responses to neurons, glia, mesentary, hepatocytes) to have a curative beneficial effect; that is they make

their way past the epithelia to reach pneumocytes to engulf pneumocytes that have viruses and cure pneumonia when they do that. So, is there a chemical peptide or protein that causes luekocyctes and other immunocytes to move past, or between, epithelial cytes twice as often, twice as fast, or even travel preferntially along tissue cytes and omit a travel path that is along epithelial lined passgaeways like capillaries? Those would be drugs that multiply the effectiveness of the immune system at interacting with and vanquishing infections. Kindof like the utility of antibiotics, it could be possible to terminate twice as many infected cytes orinvasive organismsevery 24 hours, rapidifying recovery from illness and improving wellbeing.

screenalibrary,

lookfor variants at a human population, find the genetics of passing epthelia or omitting travel at epithelial separation from the tissue cytes to be beneficially immunoterminated. Gene therapy could then produce muchhigher effectiveness immune systems at the people whoget the genetherapy who then have immune function that is twice as effective as passing, or passes twice as frequetly throughepithelia. Similarly my perceptionisthe leukocytes WBCs and other immunocytes have chemoreceptors. it is possible that at humans some human immune systems have twice the ability to sense, accurately a immunomeaningful chemical or molecule; These might be much

more effective at sensing things at a distance when to pass an epithelial structure like a capillary to reach an actual tissue cyte to terminate the deleterious cytes and cause healing. One thing supporting the twice as effective idea is that at other body systems human capability differences between and amongst 2019 AD humans ranges over amounts much more than twice as effective, human vision, from 20-15 to 20-30, at immunoreactivity, children and adults have notably different immune learning libraries, much more than two times different, even persons of normal mental capability during 2019 AD could have brains that were twice or half as weighty. The technology is then, find an area of the immune system, particularly rapid, affordable, and effective at being changed, where the difference betweena human

withthe most beneficial versionand a median activity humanis twice the effectiveness or greater. Then amongst that list of doubled or higher immune system capability, find the capabilities that respond withgreater ability most effectively to drugs, plants or other organisms, gene therapy, germline modification, lifestyle changes, and

Possibly things like biologically originated duration of immunity from an exposure could cause some people to be ill half as often (twice effectiveness) at areas, mostly previous to the 21st century, where disease organisms were persistent at the environment. So these persons would be able to omit becoming ill twice a as long or twice as effectively as others when re-exposed.

Adjuvants. I read about vaccine

adjuvants. Are there any physiologically harmless adjuvants, possibly what were known during 2019 AD as GRAS food additives? **Adjuvants similar to those that are a part of vaccines; these adjuvants when taken orally cause the entire volume of the body to be more effective at beneficially developing an immune response.** So basically, if a person gets athlete's foot, taking an oral adjuvant makes them twice as effective at developing an effective immune response to the fungi, so that makes them be cured twice as fast and be half as likely to have a recurrence. It is possible a bodywide adjuvant could reduce pneumonia, saving lives, although infection frequency could just be from organism variety; also, there is research on food, lifestyle, and cancer prevention.

I have not heard of how taking adjuvant pills could make it so **non detectable carcinogenesis is twice as immuno reacted to**, thus the persons as a population, as measured at a population, get cancer as disease half as often.

Wikipedia mentions PAMPS, “Adjuvants accomplish this task by mimicking specific sets of evolutionarily conserved molecules, so called PAMPs, which include liposomes, lipopolysaccharide (LPS), molecular cages for antigen, components of bacterial cell walls, and endocytosed nucleic acids such as double-stranded RNA (dsRNA), single-stranded DNA (ssDNA), and unmethylated CpG dinucleotide-containing DNA.[4] Because immune systems have evolved to recognize these specific antigenic moieties, the presence of an

adjuvant in conjunction with the vaccine can greatly increase the innate immune response to the antigen by augmenting the activities of dendritic cells (DCs), lymphocytes, and macrophages by mimicking a natural infection.” things that look like cytostructures, a lot of different things have as adjuvants.”, screening large libraries of PAMPS could find better adjuvants and possibly those that if taken orally, possibly as parts of liposomes, provide beneficial systemic immune sytem enhancement.

It is possible to imagine liposomes, or artificial lipid bilayer bags, that have proteins on their surfaces causing engineerable changes in immune response, that is, being adjuvants;

Bag surface proteins could be kind of purposefully noncomplex, priming for a broad different similar group of

proteins to recognize. Noting the lipid bilayer bag could be made out of different lipids it is possible lipid bags made out of really lengthy new omega 3s like C20 or C27 could be hyperdurable yet physiologically harmless or even measurably beneficial as omega-3s when they disintegrate. My perception is that some kinds of liposomes are ultraffordable to produce, sort of like: oil, water, ultrasonic transducer manufacturing.

They could screen a library of mlecules to find out if there are better adjuvants than “alum”, aluminum phosphate. Gallium is at the same row of the periodic table so they could try that. Also, noting the milligrams or possibly even micrograms of aluminum phosphate at an injection it is possible aluminum phosophate

liposomes that make it past the GI tract could produce the same chemical concentration as an injected dose from oral consumption; it could have its flavor made palatable rather than tasting like alum because of the liposomes.

Also, they could find out if oral food: pickles, which contain alum, have an immunobeneficial, all-body adjuvant effect on laboratory mammals, and also do correlation studies on humans that eat pickles. It is possible there are other adjuvant foods, possibly some kind of recipe for liposomal transport of something immunoadjuvantish, so like a salad dressing, a nugget dipping sauce, a dilute milk drink, or a new kind of quantifiably physiologically beneficial margarine to make liposomes or lipid bilayer bags with and at. There is a

chance that oil coated fried potatos like french fries could have a liposome producing oil-water effect, or a purposeful coating; the technology to think of though is what is at the core of the liposomal bag; PAMPS; something like “vegemite”, dried yogurt powder enzyme product (basically a bunch of bacterial cytostructures and mambranes as a heap of legos), I read activated carbon, and I think zeolite like aluminum oxide, so possibly some naturally occuring zeolite like mineral, at liposomal bags. They could also make artificial zeolite that is, imaginably many times more effective than other forms of aluminum oxide, although it might not be.

If zeolites work as adjuvants, then a variation of the food thing sodium silico aluminate might be sort of like a

ceramic material that could have a different zeolite like form that could be an adjuvant.

silica gel adjuvants: Other silica materials that might have adjuvant character, be placeable at liposomes, or could have an adjuvant like aluminum or gallium phosphate or ion exchange resins dissolved in them are silica gels, it is possible silica gels have different AMU amounts of silica networks, so perhaps they have light and medium ones, ones that adsorb strongly, others that adsorb weakly, also, silica gels can absorb liquids which I perceive might diffuse out again over hours, so if there is a liquid adjuvant it might go well with silica gel with liposomal bag around it; “The hydroxy (OH) groups on the surface of silica can be functionalized to afford specialty silica gels that exhibit unique

stationary phase parameters. These so-called functionalized silica gels are also used in organic synthesis and purification as insoluble reagents and scavengers.” makes custom silica gels sound like they can be tuned to absorb or adsorb custom chemicals, there is even a chance that they could do some sort of simple localization, although it seems like to a silica gel, everything they are near would be epithelium.

Another, new to me approach to an oral immunization: If silica gels that are really eentsy are placed in liposomes then migrate through the small intestine to the circulatory sytem then perhaps they meet immunocytes like leukocytes or macrophages or WBCs or some other thing that react to them as a granular particle to be engulfed, I do not know

how it works, but after being engulfed a silica gel particle might diffuse out something, even a liquid, that then causes a beneficial immune reaction.

Alginate jello blobs at liposomes might be an adjuvant, “adjuvants may provide physical protection to antigens which grants the antigen a prolonged delivery” That suggests the possibility that putting alginate jello in liposomes would cause alginate filled oil-water bags to travel around the circulatory system. These might pruprosefully leak alginate, causing some amount of antigens to get alginated-attached and then get gradually re-emitted; also **ion exchange resins could be at liposomal bags and even glom and then gradually re-emit biological molecules of particular masses and surface charges.**

Somewhat dubious, but it is my perception there are sometimes trace amounts of blood in stools. That suggests that an adjuvant, like a PAMP, aluminum or gallium phosphate at liposomes, that dissolves or migrates through membranes at the large intestine, or just contacts the sides of the large intestine, which, based on the blood at a stool concept, might have circulatory system distribution possibilities for something like the micrograms or milligrams of something like alum that could be a bodywide adjuvant.

longevity technology:

Gene therapy technology:

It seems like a dermal dose of thioglycolate based hair remover

cream that, at more minutes of activity than I think the 2019 label suggests, causes moist plasma-like exudate would be an effective, simple to dose (just calculate cm^2 dermal area to topically dose to get a particular number of milligrams or micrograms of gene product per 24 hours) gene therapy technology, then to produce even greater effectiveness, have the gene therapy modified dermis have an optically activated activateable gene or gene therapy promotor area that would make it so that you could further program and direct the dosage with laser (like blue laser pointer) activation or the application of a highly available chemical. Among these technology methods is having dermal gene therapy respond to topical materials, many of which could be available almost without effort, for example,

topical tannin, which could come from a leaf poultice available anywhere there are leaves, or a pharmacy, as the person prefers, could activate all, or just a little part, or even just an easy to remember location (poultice on your elbow once a month or once a year to omit ageing) dermal gene therapy area that causes some particular healing or also wellness response, as well as a longevity increasing, healthspan increasing, youthspan increasing, and lifespan increasing chemical, or as another item, produce a voluntary behavior enhancing chemical, a mental wellness drug, a cardiovascular well being drug, a senolytic, as well as a cognitive preserving and cognitive enhancing drug, an general immunoactivating drug that causes the production of more of the immunochemicals or immunocytes

that are the first to notice something like a nonenutral nonbeneficial organism at the body, and, as the research finds effective treatments, an anti-alheimers protein drug; along with those technologies listed and described new better drugs that are thought of, found, and made into dermally available, light or chemically activateable gene therapy forms abe beneficial and creatable. Like I will see if I can think of a new one, if I can then that verifies humans, that is persons, that is people can invent new beneficial medicines. How about fMRI drugs? fMRI of a various thoughts and emotions that get the same verbal or writing or software measured, psychology test quantifgied description while having different fMRI data graphics, which is actually a form of algorithmic math representation, as well as positron

emission tomography representation; These identically described but experientially more beneficial ways of a brain having being, isness, content, responsive ability, cognition, feeling, resting or active ambience would be different than they were before adjustable, beneficial, voluntary and even reversible or wears-off (recoverable) gene therapy; these cognitive ways, mental baselines, emotions, and even different than the thinker things like dreams or responses to media, to the extent that although they have differing fMRI or as well as positron emission tomography graphic and math measures they have identical measured quantifications at tests, computer tests, and even a person’s self-generated written descriptions of the experience of having a way of cognition, feeling, or awareness as

written by the person. Math is used to define a data space, and the data space here is, with fMRI orpositron emission tomography visual feedback there is something you can prefer, and make actual with putting a leaf poultice or light ray on your arm to make durably the preferred optimized usual way to be, that can be reversed if you like, that is undetectable to prose, 2019 AD psychology measurements, the surveyed perception of other people, measurements of productivity of any kind, or computer neural network (such as deep-learning) predictions of your behavior. You like things better, feel better, think better, emote better, and do many other things better, but it is undetectable to others. When you activate the gene therapy area, such as a light or topical material activation of an area of dermis that gene therapy

causes a shift to that version of genetics that causes the fMRI or positron emission tomography math and graphics to have software create the actual biological gene sequences, that will produce your, that is the utilizers, preferred version of the cognitive direction, ambience, and experience that is preferred. You can put some leaf poultice or shine a bright light on your elbow once a year to create your preferred form or kind of happy, your preferred kind of imaginative, and your preferred kind of productivity, orderliness or tidiness. It is the “even more opposite than a P-zombie drug” that gives humans, that is persons, that is people, the ability to experience, prefer, and activate benefits which are language, AI (without digital tomography), behavioral, productivity form and amount, as well as psychologically

surveyably really unlikely to be discernable or distiguishable, although of course fMRI or positron emission tomography of 3d (4d time) maps of neurotransmitter locations and activation is mathematically and scientifically modellable, predictable, and the source of new materials, ideas, content, and applications with math and science. It is just nifty to be able to make actual a preference of being, isness, response to things, mental forms, nonself-forms like dreams, as well as other attributes that are among the beneficial, all of these while, being absent the possibility of verbal, written, or nonmathematical description. This gene therapy is a way to be the thing, the more womderful optimal thing, you could not ever describe, after sampling it, and being able to reverse your activation of the newly optimized

way of being. Also, as an amazing feature, any human, that is person, or people, that modified themselves this way would, with very high likeliness of nondetectability, function exactly the same in the perceptions of others. You would feel creative and happy and productive, and even when between thoughts, feelings, as well as sensations prefer the newly optimized form at the new way you prefer, yet those around you would be absent noticing any change. Along with beneficial, nearer to being a preferred optimal, change that is outside of description and discernability to others, the person, that is human, or people would like to they can also get voluntary, voluntarily durable, voluntarily reversible, gene therapy that causes perceptible beneficially experienced personal voluntary change that other people can perceive

if that person, human, or people feel like it.

Is there any beneficial protein or amino acid that if gene therapy produced at the optical areas or whites of the eye would benefit human beings? I read that cataracts are treatable with some eyedrops made from an amino acid, it is possible gene therapy with one dose of instilled fluid could **prevent cataracts** or provide a one dose cure for cataracts when it causes the production of the cataract curing amino acid. it is even possible that halving or quartering the immunoreactivity of corneas and lenses using gene therapy would make them stay clearer longer with less surface variation. AEDG and the thymulin peptide combined make

humans 4 times more likely to be alive after a 6 year interval, so an eye instilled gene therapy fluid that causes the eyes to make AEDG and thymulin could possibly be a one dose longevity, wellness existence preserving functional activity drug.